

Health and Environmental Effects
Profile for N-Ethylaniline

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HEALTH AND ENVIRONMENTAL EFFECTS PROFILE
FOR N-ETHYLANILINE

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16. ABSTRACT The Health and Environmental Effects Profile for <u>N-Ethylaniline</u> was prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Solid Waste and Emergency Response to support listings of hazardous constituents of a wide range of waste streams under Section 3001 of the Resource Conservation and Recovery Act (RCRA) and to provide health-related limits for emergency actions under Section 101 of the Comprehensive Environmental Response, Compensation and Liability Act (CERCLA). Both published literature and information obtained from Agency program office files were evaluated as they pertained to potential human health, aquatic life and environmental effects of hazardous waste constituents. Quantitative estimates have been presented provided sufficient data are available. Existing data are insufficient to determine a Reference Dose (RfD) or a carcinogenic potency factor for <u>N-ethylaniline</u>. The Reportable Quantity (RQ) value of 1, 10, 100, 1000 or 5000 pounds is used to determine the quantity of a hazardous substance for which notification is required in the event of a release as specified by CERCLA based on chronic toxicity. The RQ value for <u>N-ethylaniline</u> is <u>1000</u>.		
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PREFACE

Health and Environmental Effects Profiles (HEEPs) are prepared for the Office of Solid Waste and Emergency Response by the Office of Health and Environmental Assessment. The HEEP's are intended to support listings of hazardous constituents of a wide range of waste streams under Section 3001 of the Resource Conservation and Recovery Act (RCRA), as well as to provide health-related limits for emergency actions under Section 101 of the Comprehensive Environmental Response, Compensation and Liability Act (CERCLA). Both published literature and information obtained from Agency program office files are evaluated as they pertain to potential human health, aquatic life and environmental effects of hazardous waste constituents. The literature searched and the dates of the searches are included in the section titled "Appendix: Literature Searched." The literature search material is current through November, 1985.

Quantitative estimates are presented provided sufficient data are available. For systemic toxicants, these include Reference doses (RfDs) for chronic exposures. An RfD is defined as the amount of a chemical to which humans can be exposed on a daily basis over an extended period of time (usually a lifetime) without suffering a deleterious effect. In the case of suspected carcinogens, RfDs are not estimated in this document series. Instead, a carcinogenic potency factor of q_1^* (U.S. EPA, 1980) is provided. These potency estimates are derived for both oral and inhalation exposures where possible. In addition, unit risk estimates for air and drinking water are presented based on inhalation and oral data, respectively.

Reportable quantities (RQs) based on both chronic toxicity and carcinogenicity are derived. The RQ is used to determine the quantity of a hazardous substance for which notification is required in the event of a release as specified under CERCLA. These two RQs (chronic toxicity and carcinogenicity) represent two of six scores developed (the remaining four reflect ignitability, reactivity, aquatic toxicity and acute mammalian toxicity).

The first draft of this document was prepared by Syracuse Research Corporation under EPA Contract No. 68-03-3228. The document was subsequently revised after reviews by staff within the Office of Health and Environmental Assessment: Carcinogen Assessment Group, Reproductive Effects Assessment Group, Exposure Assessment Group, and the Environmental Criteria and Assessment Office in Cincinnati.

The HEEP's will become part of the EPA RCRA and CERCLA dockets.

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EXECUTIVE SUMMARY

N-Ethylaniline is a colorless liquid with an aniline-like odor that rapidly turns brown when exposed to light and air (Northcott, 1978; Windholz, 1983). It is soluble in a variety of organic solvents but is almost insoluble in water. Currently, four companies manufacture this chemical in the United States (SRI, 1985; USITC, 1985). The information on current production volume of this chemical is unavailable, but 1.05 million pounds was produced in 1976 (Bannister et al., 1979). N-Ethylaniline is manufactured by the reaction of aniline with ethanol in the presence of a sulfuric acid catalyst at 200°C, or by alkylating aniline in the presence of sodium amide at 275°C (Rosenwald, 1978). N-Ethylaniline is used in the manufacture of dyes and as an explosive stabilizer (Northcott, 1978).

Adequate information is not available in the literature to assess the environmental fate and transport of N-ethylaniline in air, water or soil. Based on the aquatic fates of other aromatic amines, it is predicted that the two important processes that may account for the significant loss of this compound from water are photooxidation and chemical oxidation. The effects of either of these processes on the fate of N-ethylaniline have not been comprehensively studied. The overall half-life of N-ethylaniline that results from all processes in Rhine River water was reported to be 1.1 days (Zoeteman et al., 1980). This compound is not expected to bioconcentrate in aquatic organisms (Sasaki, 1978; Kawasaki, 1980). In the air, reaction with OH radical may be the most significant process for N-ethylaniline. Based on the rate constant of this reaction for similar aromatic amines, it is estimated that the half-life of this reaction will be ~2 hours. In soil, catalyzed free radical oxidation may be the most significant reaction for

this chemical (Dragun and Helling, 1985). The results of the Piet et al. (1981) study suggest that moderate biodegradation of this chemical may occur in soil. Moderate leaching of this chemical from soil to groundwater is likely (Piet et al., 1981).

Surface waters in the United States have rarely been monitored for N-ethylaniline. This compound was found in one U.S. river, and its presence was attributed to the effluents from an unspecified chemical industry (Shackelford and Keith, 1976). N-Ethylaniline has been detected in the Netherlands in the River Waal (Meijers and Van der Leer, 1976; Zoeteman et al., 1980) and the Rhine River at a concentration of 0.3 µg/l (Zoeteman et al., 1980). N-Ethylaniline has never been detected in any drinking water. Monitoring data on the level of N-ethylaniline in the atmosphere or in food could not be located in the available literature. Intravenous solutions packed in polyethylene bottles, which used sandwiched rubber discs between two polyethylene layers for the closure, contained between 0.1 and 2 mg/l of N-ethylaniline (Ulsaker and Telen, 1979).

Little information is available concerning effects of N-ethylaniline on aquatic organisms. Tonogai et al. (1982) reported 24- and 48-hour LC₅₀ values of 71 and 33 mg/l, respectively, for the medaka, Oryzias latipes. Growth of the saltwater blue-green alga, Agmenellum quadruplicatum, was inhibited by concentrations of ≥500 µg/dish in an algal lawn bioassay.

With the exception of the reports on the in vitro p- and N-hydroxylation of N-ethylaniline (Hlavica and Kiese, 1969; Hlavica, 1970; Kroeber et al., 1970), pertinent data regarding the absorption, distribution, metabolism and excretion of N-ethylaniline could not be located in the available literature as cited in the Appendix.

Pertinent data regarding the carcinogenicity, teratogenicity and reproductive effects of N-ethylaniline could not be located in the available literature as cited in the Appendix. N-Ethylaniline was not mutagenic to S. typhimurium (Florin et al., 1980). Chronic exposure of rats to 14.5 mg/kg/day of N-ethylaniline (5% of the oral LD₅₀) resulted in decreased hemoglobin and red cell counts and increased methemoglobin levels and white cell counts (Yalkut, 1971). Short-term studies indicate that N-ethylaniline caused increased methemoglobinemia in rabbits (Bayerl and Kiese, 1965) but was not toxic to the cilia of the chicken trachea in vitro (Pettersson et al., 1982).

Data were insufficient to derive an ADI or potency factors for N-ethylaniline. An RQ of 1000 was derived based on the hematological effects in rats chronically treated with an oral dose of 14.5 mg/kg/day in the study by Yalkut (1971). Because data regarding the carcinogenicity to animals and humans were lacking, N-ethylaniline was classified as an IARC Group 3 and an EPA Group D chemical.

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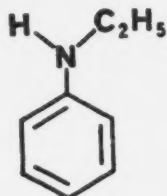
LIST OF ABBREVIATIONS

ADI	Acceptable daily intake
BCF	Bioconcentration factor
BODT	Biological oxygen demand, theoretical
CAS	Chemical Abstract Service.
CS	Composite score
K_{oc}	Soil sorption coefficient standardized with respect to organic carbon
K_{ow}	Octanol/water partition coefficient
LC ₅₀	Concentration lethal to 50% of recipients (and all other subscripted dose levels)
LD ₅₀	Dose lethal to 50% of recipients
MED	Minimum effective dose
RQ	Reportable quantity
RV _d	Dose rating value
RV _e	Effect rating value

1. INTRODUCTION

1.1. STRUCTURE AND CAS NUMBER

The chemical commonly called N-ethylaniline is also known as N-ethylbenzenamine, ethylaniline; ethylphenylamine; and N-ethylaminobenzene (SANSS, 1986). The structure, empirical formula, molecular weight and CAS Registry number of this chemical are as follows:



Empirical formula: $C_8H_{11}N$

Molecular weight: 121.18

CAS Registry number: 103-69-5

1.2. PHYSICAL AND CHEMICAL PROPERTIES

N-Ethylaniline is a colorless liquid with an aniline-like odor that rapidly turns brown when exposed to light and air (Northcott, 1978; Windholz, 1983). It is soluble in ethanol, ethyl ether and other organic solvents, but is almost insoluble in water (Windholz, 1983). Some of the physical properties of this chemical are given below.

Melting point:	-63.5°C	Windholz, 1983
Boiling point:	204.5°C	Windholz, 1983
Density:	0.958 g/cm ³ at 25/25°C	Windholz, 1983
Flash point: (open cup)	85°C	Hawley, 1981
Vapor pressure:	0.39 mm Hg at 25°C (extrapolated from higher temperature values)	Boublik et al., 1984; Chao et al., 1983
Water solubility:	177.8 mg/l (estimated)	Dragun and Helling, 1985
Log K _{ow} :	2.16 (recommended)	Hansch and Leo, 1985

N-Ethylaniline can undergo electrophilic substitution reactions. It oxidizes in air at room temperature and, like other secondary amines, it can form n-nitrosamine in the presence of nitrous acid (Gutsche and Pasto, 1975; Northcott, 1978).

1.3. PRODUCTION DATA

According to the TSCA Inventory, three companies manufactured and one company imported this chemical in the United States in 1977 (U.S. EPA, 1977). The U.S. production volume of this chemical in 1976 was 1.05 million pounds (Bannister et al., 1979). As of January 1985, the following three companies manufactured N-ethylaniline in the United States (SRI, 1985): Buffalo Color Corp., Buffalo, NY; DuPont Co., Inc., Deepwater, NJ; and First Mississippi Corp., Pascagoula, MS.

In addition, USITC (1985) reported that Atlantic Industries Inc., Nutley, NJ, currently produces this chemical in the United States; however, no data on the current production volume were available. N-Ethylaniline is commercially produced by reacting aniline with ethanol in the presence of a sulfuric acid catalyst at 200°C to produce a mixture of primary and secondary amine. Another method that results in a 75% yield of N-ethylaniline is the reaction of aniline with ethanol in the presence of sodium amide at 275°C (Rosenwald, 1978).

1.4. USE DATA

N-Ethylaniline is used as an intermediate in the manufacture of dyes and as a stabilizer for explosives (Northcott, 1978).

1.5. SUMMARY

N-Ethylaniline is a colorless liquid with an aniline-like odor that rapidly turns brown when exposed to light and air (Northcott, 1978; Windholz, 1983). It is soluble in a variety of organic solvents but is

almost insoluble in water. Currently, four companies manufacture this chemical in the United States (SRI, 1985; USITC, 1985). The current production volume of this chemical is unavailable, but 1.05 million pounds was produced in 1976 (Bannister et al., 1979). N-Ethylaniline is manufactured by the reaction of aniline with ethanol in the presence of a sulfuric acid catalyst at 200°C, or by alkylating aniline in the presence of sodium amide at 275°C (Rosenwald, 1978). N-Ethylaniline is used in the manufacture of dyes and as an explosive stabilizer (Northcott, 1978).

2. ENVIRONMENTAL FATE AND TRANSPORT PROCESSES

2.1. WATER

Limited information regarding the fate and transport of N-ethylaniline in water is available in the literature. Based on the aquatic fates of other aromatic amines (i.e., N,N-dimethyl- and N,N-diethylaniline) the two important processes that may degrade N-ethylaniline in natural waters are photooxidation and oxidation by oxygen in water. Neither of these processes has been studied comprehensively to determine their effects on N-ethylaniline in water. Using the Japanese biodegradability test, which consists of incubating 100 mg/l of the test substance with activated sludge as microbial inoculum at 25°C for 2 weeks, Kitano (1978) found that N-ethylaniline was nonbiodegradable. Compounds that consumed <30% of the BODT were deemed to be nonbiodegradable (Sasaki, 1978).

Data regarding the removal of N-ethylaniline from natural water by volatilization or sorption onto suspended particles with subsequent sedimentation could not be located in the available literature as cited in the Appendix. A qualitative prediction about the volatility of a chemical from water can be made from Henry's Law constant. Based on the vapor pressure and the solubility data given in Section 1.2., the estimated Henry's Law constant for N-ethylaniline is $3.52 \times 10^{-4} \text{ atm-m}^3 \text{ mol}^{-1}$. According to Lyman et al. (1982), a value of $3.5 \times 10^{-4} \text{ atm-m}^3 \text{ mol}^{-1}$ indicates that N-ethylaniline volatilization from water may occur but the process may not be rapid. In addition, the formation of covalently bonded compounds with humic materials in water (see equations 2-1 and 2-2) will reduce the availability of the free compound in water and subsequently

reduce the significance of the volatilization process. The K_{oc} for this chemical may be estimated from the following equations (Lyman et al., 1982):

$$\log K_{oc} = -0.55 \log S + 3.64 \quad (S \text{ is solubility in mg/l}) \quad (2-1)$$

$$\log K_{oc} = 0.544 \log K_{ow} + 1.377 \quad (2-2)$$

Using the values for solubility and K_{ow} given in Section 1.2., the $\log K_{oc}$ is estimated to be 2.40 and 2.55 from Equations 2-1 and 2-2, respectively. Therefore, it is expected that this compound will be moderately adsorbed onto particulate matter in water and partially removed from the aquatic phase by sedimentation. In addition to the sorption process, N-ethylaniline is likely to react with humic materials in suspended water particles to form covalently bonded compounds (Parris, 1980). Since this reaction is not readily reversible, the portion of aquatic N-ethylaniline that undergoes this reaction will not be available for reentry into the aquatic phase.

The overall half-life of N-ethylaniline resulting from all processes in Rhine River water was reported by Zoeteman et al. (1980). By monitoring the decay of the concentration of N-ethylaniline along the length of the river where no important discharges of chemicals were expected and assuming a first-order overall degradation, these authors estimated the half-life of N-ethylaniline as 1.1 days.

The bioconcentration of N-ethylaniline by aquatic organisms as determined by the Japanese biodegradability and bioaccumulation test was examined by Sasaki (1978) and Kawasaki (1980). Kawasaki (1980) reported a BCF value for this compound of <10 and Sasaki (1978) confirmed that this compound was "nonaccumulative or low accumulative."

2.2. AIR

Pertinent data regarding the fate and transport of N-ethylaniline in the atmosphere could not be located in the available literature as cited in the Appendix. The rate constants for OH radical reactions with aniline and N,N-dimethylaniline were reported to be 118×10^{-12} and 148×10^{-12} $\text{cm}^3 \text{ molecule}^{-1} \text{ sec}^{-1}$, respectively, at 25°C (Atkinson, 1985). If it is assumed that the concentration of OH radical in the atmosphere is 10^6 molecule cm^{-3} (Cupitt, 1980) and that the rate constant for OH radical reaction with N-ethylaniline is between 118×10^{-12} and 148×10^{-12} $\text{cm}^3 \text{ molecule}^{-1} \text{ sec}^{-1}$, the half-life for this reaction can be estimated to be ~2 hours. Therefore, this reaction is expected to be responsible for the removal of significant amounts of N-ethylaniline from the atmosphere.

Although no experimental or estimated data on the rate of removal of N-ethylaniline through atmospheric physical processes are available, based on its aqueous solubility and vapor pressure data it is anticipated that significant removal of this compound through wet deposition and absorption onto atmospheric aerosol particles and subsequent precipitation will not occur (Cupitt, 1980).

2.3. SOIL

Soil and clay-catalyzed free radical oxidation of N-ethylaniline may be the most significant reaction of this chemical in soil (Dragun and Helling, 1985). The half-life for this reaction cannot be estimated because of the lack of kinetic data on this reaction. Because of light attenuation and scattering, photooxidation in soil may not be significant beyond the superficial soil layer. Piet et al. (1981) studied the behavior of several chemicals including N-ethylaniline during infiltration through a soil layer

consisting of sand, clay and peat. At a residence time of 18 days, the concentration of N-ethylaniline/ethylmethyl pyridine (could not be separated by the analytical method used) was reduced ~80% as a result of passage through the soil bed. This reduction in concentration was probably not due to sorption alone because the compound would have attained its breakthrough in time (the soil bed was continuously used for 4 years for infiltration purposes). Since a possible explanation for the reduced concentration is biodegradation, it is likely that it will play a significant role in the removal of N-ethylaniline from certain types of soils.

Studies regarding the loss of N-ethylaniline through evaporation from soil could not be located in the available literature as cited in the Appendix. Based on its estimated volatilization potential from water (see Section 2.1.), it is predicted that volatilization from soil surfaces may not be significant. The probability of N-ethylaniline leaching through soil will depend on its K_{oc} and the likelihood of biodegradation.

From the estimated $\log K_{oc}$ of 2.40-2.55 (see Section 2.1.), it is predicted that N-ethylaniline will be moderately sorbed onto soil. Since the biodegradation of this compound in soil is expected to be moderate, some leaching into groundwater is expected. The infiltration experiments of Piet et al. (1981) showed that ~20% of the compound leached through a soil consisting of sand, clay and peat within 18 days (depth of compound in soil not specified). A significant portion of N-ethylaniline is also likely to react with humic materials in the soil to form covalently bonded amino-substituted quinones (Parris, 1980) and as a result may become nonleachable in soil. In experiments with N-methylaniline and humic acids, Parris (1980) reported that depending on the nature of the humic acids, ~40-54% of the compound

became immobile in 8 days as a result of this covalent reaction. Therefore, a significant amount of N-ethylaniline will be rendered nonleachable in soil because of this covalent reaction.

2.4. SUMMARY

Adequate information is not available in the literature to assess the environmental fate and transport of N-ethylaniline in air, water or soil. Based on the aquatic fates of other aromatic amines, it is predicted that the two important processes that may account for the significant loss of this compound from water are photooxidation and chemical oxidation. The effects of either of these processes on the fate of N-ethylaniline have not been comprehensively studied. The overall half-life of N-ethylaniline from all processes in Rhine River water was reported to be 1.1 days (Zoeteman et al., 1980). This compound is not expected to bioconcentrate in aquatic organisms (Sasaki, 1978; Kawasaki; 1980). In the air, reaction with OH radical may be the most significant process for N-ethylaniline. Based on the rate constant of this reaction for similar aromatic amines, it is estimated that the half-life of this reaction will be ~2 hours. In soil, catalyzed free radical oxidation may be the most significant reaction for this chemical (Dragun and Helling, 1985). The results of the Piet et al. (1981) study suggest that moderate biodegradation of this chemical may occur in soil. Moderate leaching of this chemical from soil to groundwater is likely (Piet et al., 1981).

3. EXPOSURE

3.1. WATER

The likely sources of N-ethylaniline in surface waters are effluents from the manufacturing and user sites of this chemical, wastewaters from oil shale processing plants and coal liquefaction operations (Slevers et al., 1981; Herbes et al., 1983). Surface waters in the United States, however, have rarely been monitored for this chemical. The presence of this compound in the Pacolet and Enoree River, SC, was reported by Shackelford and Keith (1976). The source of the compound in the river was the effluent from an unspecified chemical industry. N-Ethylaniline has been qualitatively identified in water from the River Waal at Brakel in the Netherlands (Meijers and Van der Leer, 1976). This compound was detected at a concentration of 0.3 $\mu\text{g}/\text{l}$ in water from the Rhine River at Lobith in the Netherlands (Zoeteman et al., 1980). The presence of N-ethylaniline in drinking water in the United States or elsewhere in the world has not been reported.

3.2. AIR

Pertinent data regarding the atmospheric level of this compound could not be located in the available literature as cited in the Appendix.

3.3. FOOD

Pertinent data regarding the level of this compound in foods could not be located in the available literature as cited in the Appendix. Ulsaker and Teien (1979) reported that N-ethylaniline was present in intravenous solutions packed in polyethylene bottles. The source of N-ethylaniline was the rubber disc that was sandwiched between two polyethylene layers used for the closure of the bottle. Depending on the content of the intravenous solution, the measured concentration of N-ethylaniline varied from 0.1-2 mg/l (Ulsaker and Teien, 1979).

3.4. SUMMARY

Surface waters in the United States have rarely been monitored for N-ethylaniline. This compound was found in one U.S. river, and its presence was attributed to the effluents from an unspecified chemical industry (Shackelford and Keith, 1976). N-Ethylaniline has been detected in the Netherlands in the River Waal (Meijers and Van der Leer, 1976; Zoeteman et al., 1980) and the Rhine River at a concentration of 0.3 $\mu\text{g}/\text{l}$ (Zoeteman et al., 1980). N-Ethylaniline has never been detected in any drinking water. No monitoring data on the level of N-ethylaniline in the atmosphere or in food were located in the available literature. Intravenous solutions packed in polyethylene bottles, which used sandwiched rubber discs between two polyethylene layers for the closure, contained between 0.1 and 2 mg/l of N-ethylaniline (Ulsaker and Teien, 1979).

4. PHARMACOKINETICS

4.1. ABSORPTION

Pertinent data regarding the absorption of N-ethylaniline could not be located in the available literature as cited in the Appendix.

4.2. DISTRIBUTION

Pertinent data regarding the distribution of N-ethylaniline could not be located in the available literature as cited in the Appendix.

4.3. METABOLISM

Based on several in vitro studies with microsomal enzymes, N-ethylaniline appears to undergo N-hydroxylation to produce phenylhydroxylamine and p-hydroxylation to produce p-hydroxy-N-ethylaniline (Hlavica and Kiese, 1969; Hlavica, 1970; Appel et al., 1965; Kroeber et al., 1970). Appel et al. (1965) reported species differences in the in vitro hydroxylation. The rate of N- and p-hydroxylation was about equal when liver microsomes from guinea pigs and rabbits were used, while lower rates were obtained with cat microsomes. p-Hydroxylation by microsomes from rabbits, guinea pigs, dogs and rats was inhibited by carbon monoxide. N-Hydroxylation by microsomes from rats, but not dogs and guinea pigs, was inhibited by carbon monoxide, while N-hydroxylation by rabbit microsomes was stimulated by carbon monoxide. Heinze et al. (1970) reported that microsomes from livers and corpora lutea of pigs were equally active in N-hydroxylating N-ethylaniline, while other tissues and organs were ≤ 10 -fold less active.

Pertinent data regarding the in vivo metabolism of N-ethylaniline could not be located in the available literature as cited in the Appendix.

4.4. EXCRETION

Pertinent data regarding the excretion of N-ethylaniline could not be located in the available literature as cited in the Appendix.

4.5. SUMMARY

With the exception of the reports on the in vitro p- and N-hydroxylation of N-ethylaniline (Hlavica and Klese, 1969; Hlavica, 1970; Kroeber et al., 1970), pertinent data regarding the absorption, distribution, metabolism and/or excretion of N-ethylaniline could not be located in the available literature as cited in the Appendix.

5. EFFECTS

5.1. CARCINOGENICITY

Pertinent data regarding the carcinogenicity of N-ethylaniline could not be located in the available literature as cited in the Appendix. This chemical is not scheduled for testing by the National Toxicology Program (NTP, 1986).

5.2. MUTAGENICITY

N-Ethylaniline was not mutagenic to Salmonella typhimurium strains TA98, TA100, TA1535 or TA1537 at a concentration of 3 μ mol/plate either with or without metabolic activation (Florin et al., 1980).

5.3. TERATOGENICITY

Pertinent data regarding the teratogenicity and developmental effects of N-ethylaniline could not be located in the available literature as cited in the Appendix. An abstract of a Russian study (Napalkov, 1971) reported that transplacental exposure to ethylaniline (specific location of the ethyl group not specified) was blastomogenic, which was defined as either resulting in subsequent tumor formation or increased sensitivity to subsequent exposure to tumor-causing chemicals.

5.4. OTHER REPRODUCTIVE EFFECTS

Pertinent data regarding other reproductive effects of N-ethylaniline could not be located in the available literature as cited in the Appendix.

5.5. CHRONIC AND SUBCHRONIC TOXICITY

In the only study available regarding the chronic and subchronic toxicity of N-ethylaniline, Yalkut (1971) reported a decrease in hemoglobin and red blood cell levels, as well as an increase in methemoglobin and white blood cell levels in rats treated chronically with 5% of the oral LD₅₀ of 290 mg/kg (14.5 mg/kg/day). No additional details were provided.

5.6. OTHER RELEVANT INFORMATION

The available LD₅₀ values are listed in Table 5-1. N-Ethylaniline was not toxic to the cilia of the chicken trachea in vitro (Pettersson et al., 1982). An intravenous dose of 100 mg/kg of N-ethylaniline in rabbits produced a similar increase in the methemoglobin concentration as an intravenous dose of 200 mg/kg of aniline (Bayerl and Kiese, 1965).

5.7. SUMMARY

Pertinent data regarding the carcinogenicity, teratogenicity and reproductive effects of N-ethylaniline could not be located in the available literature as cited in the Appendix. N-Ethylaniline was not mutagenic to S. typhimurium (Florin et al., 1980). Chronic exposure of rats to 5% the oral LD₅₀ of N-ethylaniline, 14.5 mg/kg/day, resulted in decreased hemoglobin and red cell counts and increased methemoglobin levels and white cell counts (Yalkut, 1971). Short-term studies indicate that N-ethylaniline caused increased methemoglobinemia in rabbits (Bayerl and Kiese, 1965) but was not toxic to the cilia of the chicken trachea in vitro (Pettersson et al., 1982).

TABLE 5-1
LD₅₀ Values for N-Ethylaniline

Route	Species	LD ₅₀ Value (mg/kg)	Reference
Oral	rat	290	Yalkut, 1971
Oral	rat	334	NIOSH, 1986
Oral	mouse	500	Yalkut, 1971
Dermal	rat	4700	NIOSH, 1986
Intraperitoneal	rat	180	NIOSH, 1986

6. AQUATIC TOXICITY

6.1. ACUTE

Tonogai et al. (1982) reported 24- and 48-hour LC_{50} values of 71 and 33 mg/l, respectively, for the medaka, Oryzias latipes.

6.2. CHRONIC

Pertinent data regarding the chronic toxicity of N-ethylaniline to aquatic organisms could not be located in the available literature as cited in the Appendix.

6.3. PLANTS

Using the algal lawn technique, Batterton et al. (1978) found that growth of the marine blue-green alga, Agmenellum quadruplicatum, was inhibited by 500 and 1000 μ g/dish but not by ≤ 100 μ g/dish.

6.4. RESIDUES

Pertinent data regarding residues of N-ethylaniline in aquatic biota could not be located in the available literature as cited in the Appendix.

6.5. SUMMARY

Little information is available concerning effects of N-ethylaniline on aquatic organisms. Tonogai et al. (1982) reported 24- and 48-hour LC_{50} values of 71 and 33 mg/l, respectively, for the medaka, Oryzias latipes. Growth of the saltwater blue-green alga, Agmenellum quadruplicatum, was inhibited by concentrations of ≥ 500 μ g/dish in an algal lawn bioassay.

7. EXISTING GUIDELINES AND STANDARDS

7.1. HUMAN

Pertinent guidelines and standards, including EPA ambient water and air quality criteria, drinking water standards, FAO/WHO ADIs, EPA or FDA tolerances for raw agricultural commodities or foods, and ACGIH, NIOSH or OSHA occupational exposure limits could not be located in the available literature as cited in the Appendix.

7.2. AQUATIC

Guidelines and standards for the protection of aquatic biota from the effects of N-ethylaniline could not be located in the available literature as cited in the Appendix.

8. RISK ASSESSMENT

Pertinent data regarding the carcinogenicity, teratogenicity and reproductive effects of N-ethylaniline could not be located in the available literature as cited in the Appendix. N-Ethylaniline was not mutagenic in S. typhimurium with or without metabolic activation (Florin et al., 1980). Chronic exposure of rats to 5% of the oral LD₅₀ of 290 mg/kg N-ethylaniline (14.5 mg/kg/day) resulted in decreased hemoglobin and red cell counts and increased methemoglobin levels and white cell counts, but lack of additional details in this study (Yalkut, 1971) precludes its use in quantitative risk assessment. Short-term studies indicate that N-ethylaniline caused increased methemoglobin levels in rabbits (Bayerl and Kiese, 1965).

9. REPORTABLE QUANTITIES

9.1. REPORTABLE QUANTITY (RQ) RANKING BASED ON CHRONIC TOXICITY

The only information on the effects of chronic and subchronic exposure to N-ethylaniline was obtained from a Russian study (Table 9-1) that reported decreased hemoglobin and red blood cell levels and increased methemoglobin and white blood cell levels in rats chronically exposed to 5% of the oral LD_{50} of 290 mg/kg of N-ethylaniline (Yalkut, 1971). Although further details on the strain and number of rats used, the actual duration of treatment, the method of administration and the levels of hemoglobin and blood cells were not provided, an RQ can be derived assuming that a dose of 14.5 mg/kg/day of N-ethylaniline resulted in adverse hematological effects. The chronic human MED of 174 mg/day was derived by multiplying the administered dose of 14.5 mg/kg/day by the cube root of the ratio of animal to human body weight, assumed to be 0.35 kg for a rat and 70 kg for a human, and by the reference human body weight (Table 9-2). A human MED of 174 mg/day corresponds to a RV_d of 2.1. The adverse hematological effects, in the absence of any additional details on the condition of the experimental animals, was assigned an RV_e of 4. The CS, which is the product of the RV_e and the RV_d , is 8.4. The corresponding RQ is 1000, as indicated in Tables 9-2 and 9-3.

9.2. WEIGHT OF EVIDENCE AND POTENCY FACTOR ($F=1/ED_{10}$) FOR CARCINOGENICITY

Pertinent data regarding the carcinogenicity of N-ethylaniline to animals or humans could not be located in the available literature as cited in the Appendix. Therefore, N-ethylaniline is a Group 3 Chemical according to the classification scheme of IARC and a Group D Chemical according to the classification scheme of EPA (U.S. EPA, 1986).

TABLE 9-1

Oral Toxicity Summary for N-Ethylaniline in the Rat^{a,b}

Exposure (mg/kg/day)	Transformed Animal Dose (mg/kg/day)	Response
14.5 "chronic"	14.5	Decreased hemoglobin and red blood cell levels; increased methemoglobin and white cell levels

^aSource: Yalkut, 1971^bSex, no. at start, average weight, vehicle/physical state and purity of compound were not reported.

TABLE 9-2

Oral Composite Scores for N-Ethylaniline in the Rat*

Animal Dose (mg/kg/day)	Chronic Human MED (mg/day)	RV _d	Effect	RV _e	CS	RQ
14.5	174	2.1	Decreased hemoglobin and red blood cell levels; increased methemoglobin and white blood cell levels	4	8.4	1000

*Source: Yalkut, 1971

TABLE 9-3

N-Ethylaniline

Minimum Effective Dose (MED) and Reportable Quantity (RQ)

Route:	oral
Dose*:	174 mg/day
Effect:	decreased hemoglobin and red blood cell levels; increased methemoglobin and white blood cell levels
Reference:	Yalkut, 1971
RV _d :	2.1
RV _e :	4.0
Composite Score:	8.4
RQ:	1000

*Equivalent human dose

10. REFERENCES

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APPENDIX
LITERATURE SEARCHED

This profile is based on data identified by computerized literature searches of the following:

GLOBAL
TSCATS
CASR online (U.S. EPA Chemical Activities Status Report)
CAS online STN International
TOXLINE
TOXBACK 76
TOXBACK 65
RTECS
OHM TADS
STORET
SRC Environmental Fate Data Bases
SANSS
AQUIRE
TSCAPP
NTIS
Federal Register

These searches were conducted in April, 1986. In addition, hand searches were made of Chemical Abstracts (Collective Indices 6 and 7), and the following secondary sources were reviewed:

ACGIH (American Conference of Governmental Industrial Hygienists). 1986. Documentation of the Threshold Limit Values and Biological Exposure Indices, 5th ed. Cincinnati, OH.

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Windholz, M., Ed. 1983. The Merck Index, 10th ed. Merck and Co., Inc., Rahway, NJ.

In addition, approximately 30 compendia of aquatic toxicity data were reviewed, including the following:

Battelle's Columbus Laboratories. 1971. Water Quality Criteria Data Book. Volume 3. Effects of Chemicals on Aquatic Life. Selected Data from the Literature through 1968. Prepared for the U.S. EPA under Contract No. 68-01-0007. Washington, DC.

Johnson, W.W. and M.T. Finley. 1980. Handbook of Acute Toxicity of Chemicals to Fish and Aquatic Invertebrates. Summaries of Toxicity Tests Conducted at Columbia National Fisheries Research Laboratory. 1965-1978. U.S. Dept. Interior, Fish and Wildlife Serv. Res. Publ. 137, Washington, DC.

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